Posterior fossa subdural hematoma of the newborn

Case report

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A newborn infant with a posterior fossa subdural hematoma is described, and nine similar cases from the literature summarized. A postnatal asymptomatic period was followed by signs of increased intracranial pressure. The diagnosis was established on the basis of negative subdural taps, bloody or xanthochromic ventricular fluid under increased pressure, and demonstration of a posterior fossa mass on the ventriculogram. Surgical evacuation with careful observation for an associated intracerebellar hematoma is the treatment of choice. Five of the 10 cases developed postoperative communicating hydrocephalus.

Key Words: subdural hematoma · posterior fossa · hydrocephalus · newborn · intracranial hypertension

Hemorrhage in the posterior fossa of newborn infants is not an unusual finding at autopsy. Such hemorrhages are usually massive and due to rupture of the vein of Galen or tentorial tears into the lateral or straight sinus, probably secondary to head molding.5,7 A rare but more important lesion is the smaller treatable posterior fossa subdural hematoma of the newborn.

Matson2 focused attention on this entity and referred to unpublished cases of his own, although he stated it is quite rare. We have found nine cases described in the literature.1,4–6,8–10 The following case report and summary of the literature are presented to define the clinical problem more clearly.

Case Report

On January 19, 1969, this 7 lb 3 oz boy was born to a primiparous mother. Labor and delivery were reported as not unduly difficult. Twelve hours later respiratory distress occurred and persisted for the next week. At 10 days of age the head size was increasing; sutures were separated, and a right sixth nerve palsy was present. Following negative subdural taps, a ventriculogram demonstrated a possible posterior fossa mass with egress of air from the ventricular system, but no air above the tentorial notch. The ventricular fluid was "cherry red," under pressure of 390 to 420 mm H2O with 1460 mg% protein. Continuous ventricular drainage was instituted; the patient improved and began taking oral feedings. Six days after removal of the ventricular drain, projectile vomiting occurred, and the fontanel was again tense. Repeat ventricular tap yielded xanthochromic fluid, and a ventriculogram confirmed the impression of a posterior fossa mass (Fig. 1). Coagulation studies were normal.

Operation. On February 14, 1969, poste-
L. Philip Carter and Hal W. Pittman

gram. Through an occipital burr hole he evacuated 25 to 30 cc of dark reddish-brown fluid. The patient made a complete recovery. Subsequent cases, including ours, have presented a strikingly similar picture although the results have not always been as gratifying (Table 1).

The infants usually did well for several hours to as long as 5 days before signs of brain stem compression and increased intracranial pressure developed. This symptom-free period following the trauma of birth probably is the time required for the expanding hematoma to reach significant proportions.

The clinical picture is similar to the common convexity subdural hematoma, and in most cases subdural taps were attempted and were negative. In all cases in which the spinal or ventricular fluid was described, the fluid was bloody or xanthochromic. Ventriculography invariably made possible the diagnosis of posterior fossa mass with the usual appearance of obstructive hydrocephalus. When the aqueduct and fourth ventricle were visualized they were displaced anteriorly and superiorly and were deformed.

An interesting complication was the presence of communicating hydrocephalus following the removal of the hematoma in five of the 10 cases. Reigh and Nelson\(^9\) and Norlén, et al.,\(^9\) attributed this to blood in the subarachnoid space. In our case, markedly elevated ventricular protein and tentorial subarachnoid obstruction may have contributed to the postoperative hydrocephalus.

The site of hemorrhage is usually undetermined. Most authors felt that a small tentorial tear into a dural sinus or a ruptured bridging vein from the cerebellar surface was the origin of bleeding. Rothballer\(^9\) and Schreiber,\(^10\) however, described intracerebellar hematomas from contused cerebellum which had extruded into the subdural space.

In the majority of cases, the results were considered good even though the diagnosis and subsequent surgery frequently were delayed several weeks. There was one postoperative death due to an unrecognized associated intracerebellar hematoma.\(^10\) Our case has had multiple shunt complications resulting in inadequately controlled communicating hydrocephalus and retardation. Careful observation for associated intracerebellar

Fig. 1. Ventriculogram showing the fourth ventricle deformed and displaced anterosuperiorly.

Postoperative Course. The head circumference continued to increase, and a pneumoencephalogram demonstrated further increase in ventricular size. Although the posterior fossa and aqueductal deformity persisted 2 weeks after surgery, there was no clinical evidence of brain stem compression. A ventriculoatrial shunt was therefore performed. The shunt has been revised on several occasions, and the patient's mental development is retarded.

Discussion

In 1940, Coblentz\(^1\) first described a surgically treated posterior fossa subdural hematoma of the newborn; he made the preoperative diagnosis on the basis of forcep marks, bloody spinal fluid with xanthochromic supernatant, negative subdural taps, and a posterior fossa mass revealed by ventriculogram.
Posterior fossa subdural hematoma of the newborn

### TABLE 1
Summary of cases

<table>
<thead>
<tr>
<th>Author</th>
<th>Delivery</th>
<th>Onset of Symptoms</th>
<th>Symptoms &amp; Signs</th>
<th>Spinal or Vent. Fluid</th>
<th>Surgical Findings</th>
<th>Commun. Hydroc.</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Coblenz (1940)</td>
<td>low forceps</td>
<td>4 days</td>
<td>enlarged head, tight fontanel</td>
<td>spinal fluid bloody, vent. fluid xanthochromatic</td>
<td>25-30 cc dark reddish-brown fluid</td>
<td>no</td>
<td>excellent</td>
</tr>
<tr>
<td>T. Y. Nelson (1959)</td>
<td>breech</td>
<td>12 hrs</td>
<td>vomiting, irregular respirations, left facial palsy, tight fontanel</td>
<td>spinal fluid bloody, vent. clear</td>
<td>&quot;large amount of old semi-solid blood&quot;</td>
<td>yes</td>
<td>good</td>
</tr>
<tr>
<td>Reigh and Nelson (1962)</td>
<td>breech</td>
<td>10 hrs</td>
<td>vomiting, irregular respirations, tense fontanel, nystagmus, high pitched cry</td>
<td>spinal fluid bloody, became xanthochromatic</td>
<td>current jelly clot 3 x 4 cm, no membrane</td>
<td>no</td>
<td>excellent</td>
</tr>
<tr>
<td>Rothballer (1962)</td>
<td>breech forceps, internal &amp; external manipulation</td>
<td>&quot;next morning&quot;</td>
<td>bloody discharge from pharynx, hypotonia, tense fontanel, icterus, weak cry, slow respirations, coma</td>
<td>spinal fluid bloody</td>
<td>thin layer of subdural with contused cerebellum &amp; intra-cerebellar clot</td>
<td>no</td>
<td>retarded</td>
</tr>
<tr>
<td>Schreiber Case 1 (1963)</td>
<td>forceps</td>
<td>birth</td>
<td>limp, poor respirations, full fontanel, hypertonia, nystagmus</td>
<td>spinal fluid blood stained, vent. xanthochromatic</td>
<td>&quot;gross brown blood&quot;</td>
<td>no</td>
<td>excellent</td>
</tr>
<tr>
<td>Schreiber Case 2 (1963)</td>
<td>—</td>
<td>less than 4 days</td>
<td>—</td>
<td>spinal fluid blood stained</td>
<td>large hematoma between tentorium, lateral post. fossa, &amp; cerebellar hemisphere</td>
<td>yes</td>
<td>autopsy: hematoma in cerebellum</td>
</tr>
<tr>
<td>Schreiber Case 5 (1963)</td>
<td>“with difficulty”</td>
<td>5 days</td>
<td>&quot;collapsed,&quot; tense fontanel</td>
<td>vent. xanthochromatic</td>
<td>old blood, fluid blood, &amp; subdural clot</td>
<td>no</td>
<td>satisfactory</td>
</tr>
<tr>
<td>Norlén, et al., (1964)</td>
<td>amniotic fluid stained</td>
<td>birth</td>
<td>irregular respirations, seizures, emesis, whining cry, hypertonia, increasing head size</td>
<td>—</td>
<td>encapsulated, organized hematoma</td>
<td>yes</td>
<td>satisfactory</td>
</tr>
<tr>
<td>Pityk, et al., (1967)</td>
<td>outlet forceps</td>
<td>16 hrs</td>
<td>lethargy, emesis, hypotonia, slow respirations</td>
<td>—</td>
<td>15 ml of dark clot</td>
<td>yes</td>
<td>satisfactory</td>
</tr>
<tr>
<td>Carter and Pittman (1971)</td>
<td>outlet forceps</td>
<td>12 hrs</td>
<td>irregular respirations, emesis, lethargy, tense fontanel, icterus, 6th nerve palsy, hypotonia</td>
<td>vent. bloody, became xanthochromatic</td>
<td>15-20 cc of dark amber fluid with membranes</td>
<td>yes</td>
<td>retarded</td>
</tr>
</tbody>
</table>

hematoma at surgery as well as communicating hydrocephalus postoperatively is obviously important in the management of these cases.

**Summary**

A case of posterior fossa subdural hematoma of the newborn has been described, and nine reported cases of the entity reviewed. Signs of acute hydrocephalus and posterior fossa mass occurred, usually following a brief latent period. The diagnosis was made from negative subdural taps, bloody or xanthochromic ventricular fluid, and ventriculographic evidence of posterior fossa mass. Following evacuation of the hematoma, communicating hydrocephalus frequently necessitated shunting procedures. Although rare, posterior fossa subdural hematoma represents a curable cause of hydrocephalus and again demonstrates the importance of definitive diagnostic procedures prior to shunting.

**Acknowledgment**

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References

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